Fisher, E. R., et al., "Cigarette Smoking and Cholesterol Atherosclerosis of Rabbits," Archives of Pathology 93: 418-421, 1974.

> ". . .a causal role of cigarette smoking to arteriosclerotic heart disease has not been convincingly demonstrated."
>
> p. 418

> "The results of the present study. . .fail to reveal any adverse effect of cigarette smoking on the structural integrity of the cardiovascular system in the rabbit with and without induced atherosclerosis. . . ."

> "Although several studies have claimed that nicotine may augment the atherosclerotic process in this species [rabbit] it is noteworthy that the daily dose of this agent that was employed was equivalent to approximately 175 to 525 cigarettes per day in man; certainly an excessive and unrealistic amount."
>
> p. 421

Helmers, C., "Short and Long-Term Prognostic Indices in Acute Myocardial Infarction. A Study of 606 Patients Initially Treated in a Coronary Care Unit." Acta Medica Scandinavica (Suppl.) 555: 86, 1974.

> "A history of smoking was no more common in those who died during the first day or during the total hospital period. . . . No impaired STP (short term prognosis) has been noted in smokers as compared to non-smokers. . . . The present results were in agreement with these findings. . . . Smoking was no more common in patients who died than in survivors. . . . A history of smoking was more common among the survivors than among the deceased. . . . In the present study none of these factors (among them smoking) was of significant additional prognostic value, when the most important factors were simultaneously taken into account."

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Corday, Eliot & Stephen Richard Corday, "Prevention of Heart Disease by Control of Risk Factors: The Time Has Come to Face the Facts," The American Journal of Cardiology 35(2): 330-333, February, 1975.

> "[S]egments of research information have often been pulled together prematurely and the population propagandized into believing that the problem of arteriosclerosis has been conquered. Possibly these tactics were used to motivate Americans to practice a more prudent way of life. But we are alarmed that some national health planners have indicated that there is no need for more research on arteriosclerosis, that if a few simple rules for risk factor intervention are followed, a health problem that kills more than 1 million Americans each year will disappear."
>
> p. 330

> "[L]ife expectancy remained relatively the same because of the rising incidenc [sic] of our two main killers, arteriosclerotic heart disease and cancer. This rise in mortality from heart disease should be attributed not solely to a change in life style, but also to the fact that people are now living into the age group in which pathologic lesions and symptoms of arteriosclerosis become clinically evident."
>
> p. 331

> "It is evident that heredity is a most important factor in the ideology [sic etiology] of arteriosclerosis."
>
> p. 332

> "[T]here is no conclusive evidence that preventive measures based on correction of single risk factors will prevent arteriosclerotic heart disease."
>
> p. 332

> "It appears that our nation's overconfidence in present risk factor concepts [of atherosclerosis] is impeding development of other promising preventive approaches."
>
> p. 333

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Seltzer, C. C., "Smoking and Coronary Heart Disease in the Elderly," American Journal of the Medical Sciences 269(3): 309-315, May-June, 1975.

> "Age-standardized CHD rates and mortality ratios have been computed from data available in four major prospective cohort investigations of smoking and health. The data examined gave consistent results. For elderly men, there were no appreciable excess of risks of CHD mortality or morbidity among cigarette smokers compared to ex-cigarette smokers and non-cigarette smokers. For elderly women, the CHD rates seemed lower in continuing cigarette smokers than in ex-cigarette smokers. These results obtained from cohort data are concordant with previous analyses of secular data. Among elderly people, the risk of CHD is essentially the same with persistence of cigarette smoking than with its cessation."
>
> p. 309

Seltzer, C. C., "Smoking and Cardiovascular Disease," American Heart Journal 90(1): 125-126, July, 1975.

> ". . .the self-selected group of people who choose to smoke are also more vulnerable to CHD than nonsmokers. This hypothesis is supported by evidence indicating that smokers and nonsmokers differ in morphology, physiology, biochemistry, personality, and way of life."
>
> p. 125

Eskwith, Irwin S., "Etiology of Atherosclerotic Heart Disease," American Heart Journal 90(6): 809-810, December, 1975.

> "Over the years, physicians have assumed many diseases to be caused by tobacco. Had they been true, the cities and towns in America today would be desolate."
>
> p. 809

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Eskwith, Irwin S., "Etiology of Atherosclerotic Heart Disease," American Heart Journal 90(6): 809-810, December, 1975.

> "Our primary need in cardiology today is a few heretics who will abandon the practice of equating statistical association with the etiology of atherosclerotic heart disease. This would mean a greater emphasis on the causation of atherosclerosis itself. We have had enough of the 25-year-old chorus constantly urging the treatment of "risk" factors which so far have led to no accompanying reduction in mortality from coronary artery disease."
>
> P. 810

Blumenthal, H. T. & M. Alex, "Special Issue on Athero-Arteriosclerosis," Gerontologia 21(3): 129-76, 1975.

> "We are currently in a period of mounting crusades against cancer and heart attacks in which the impression is being created that all biomedical scientists are in accord in respect to the direction of future research. The pressure to conform follows from the fact that the funding agencies of government are appropriating large sums for research in these areas, while reducing the support for other spheres of health research. Perhaps it is relevant to note that several centuries ago it was pointed out that if one acts for long enough as though one believes, the grace of faith will eventually be given. We appear to be in a period when many biomedical scientists feel a need to behave as devout believers if they are to merit research support."
>
> p. 135

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Blumenthal, H. T. & M. Alex, "Special Issue on Athero-Arteriosclerosis," Gerontologia 21(3): 129-76, 1975.

> "[A]thero-arteriosclerosis has been transferred from the category of aging to one of disease, even though the recent Task Force on Arteriosclerosis report confesses that the basic etiology of this disorder remains elusive. Perhaps the elimination of athero-arteriosclerosis from the category of aging has been premature, since chronological age appears to provide the best correlate with this disorder."
>
> p. 144

> "For over two decades we have been subjected to popular campaigns to reduce calorie consumption, to give up cigarette smoking, to substitute polyunsaturated for saturated fats, to reduce life's tensions, and to exercise adequately. The benefits of such campaigns, judging from mortality rates and life expectancy data, are hardly spectacular."
>
> p. 144

Syme, S. L., et al., "Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii and California: Introduction," American Journal of Epidemiology 102(6): 477-480, 1975.

> "It has been observed that among men of Japanese ancestry, there is a gradient in CHD mortality increasing from Japan to Hawaii to California. . . ."
>
> p. 477

> ". . .if the incidence rates from this study confirm that differences in serum cholesterol blood pressure, and smoking do not account for the higher rates of CHD seen among Japanese migrants to the continental United States, then this should provide valuable new insights into the epidemiology of coronary heart disease. Further analyses from this study will pursue the possible effects of obesity and of cultural and dietary change on changing rates of this disease."
>
> p. 480

2026259309

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Smith, B., "Cancer Incidence," New Scientist, pp. 304-305, February 5, 1976.

> "There is a current practice by the anti-smoking lobby, of taking a multifactorial condition such as coronary occlusion or bronchitis, in which smoking may be one factor, and citing it as the primary cause. Figures currently given by Dr. Owen [U.K. Minister of Health] of the annual mortality due to smoking must use this technique. They are too large to be real. After all, half the adult population does not smoke."
>
> p. 305

Master, Kenneth, "Carbon Monoxide, Atherosclerosis, and Natives of the Highlands of New Guinea," New England Journal of Medicine 294(10): 557, March 4, 1976.

> "[E]xposure to elevated levels of carbon monoxide does not induce arteriosclerotic vascular disease in natives of the highlands of New Guinea. It is likely that inhalation of carbon monoxide alone does not directly cause atherosclerosis in man but, in combination with dietary, stress, hereditary, and other unknown factors, may predispose to it."

Whereat, Arthur F., "Prevention of Heart Disease-I," The American Journal of Cardiology 37: 449-450, March 4, 1976.

> "Twenty years ago we took the fat from our patients' diets. Ten years later that was abandoned and we tortured them by telling them to eliminate carbohydrate. We also administered drugs that caused rashes, depilated their scalps, caused them to bleed or led to obstipation; at the same time we deplored the request of the Food and Drug Administration that clinical effectiveness be documented. . . . We cannot afford to waste any more decimillions on 'comprehensive programs' for studying large populations to search for 'statistical correlations'. If the past 30 years have not taught us the lack of wisdom of such expensive projects and surveys, we will have another decade of useless correlations and precious little fundamental knowledge."
>
> p. 449

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Horgan, John H., "Coronary Heart Disease - Whither Bypass Surgery?," Journal of the Irish Medical Association 69(13): 357-361, August 21, 1976.

> "Consideration of the three major risk factors - namely, hyperlipidaemia, cigarette smoking and hypertension - reveals no evidence in man to support the contention that the control of elevated lipids, cessation of cigarette smoking or the control of hypertension retards the development of the atherosclerotic process or delays the rate at which coronary artery stenoses develop once these processes are established."
>
> p. 357

Aviado, D. M., "Chemical Factors in the Etiology of Coronary Heart Disease," Journal of the Phillipine Medical Association 52: 41-48, 1976.

> "The association of a high incidence for a particular group exposed to a medication or habit does not mean a cause-and-effect relationship. Cigarettes, coffee, oral hypoglycemics and oral contraceptives are still in use because there is no definitive proof that any of these consummable [sic] products causes coronary heart disease."
>
> p. 46

> "[C]onstitutional and other genetic factors are largely responsible for the 24-fold difference in incidence of coronary heart disease between the United States and the Phillipines."
>
> p. 46

> "[M]easurements in the canine and human heart have failed to show coronary insufficiency induced by nicotine. To the contrary, there is an increase in coronary blood flow proportionate to the demand for oxygen in the heart."
>
> p. 47

2026259311

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Gutstein, W. H., et al., "Neural Factors in Atherogenesis," Ann. N.Y. Acad. of Sci. 275: 117-130, 1976.

> "The vast majority of individuals destined to develop and die from atherosclerotic disease do so for as yet unknown reasons.
>
> "[R]ecent evidence indicates that behavioral responses and personality traits are closely correlated with the subsequent development of hyperlipidemia and IHD."
>
> p. 127

Hine, Charles H., Clinical Professor of Pharmacology and Preventive Medicine, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "[Cardiovascular responses to nicotine] are of limited duration in time and to the extent that they can be activated by the nicotine in cigarette smoke, last only for a moment."
>
> p. 135

Seltzer, Carl C., Senior Research Associate, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "There are inconsistencies in the alleged dose-response relationship of increased rates of CHD with increased amounts of cigarette smoking. The evidence shows no consistent positive gradient of CHD with amount of cigarettes smoked in a number of studies, and inversions or inconsistencies in the alleged gradient in other studies. This is particularly evident in the data from the Framingham Heart Study of the U.S. Public Health Service."
>
> p. 78

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Clayton, J. K., et al., "Effect of Cigarette Smoking on Thromboembolism," British Medical Journal, p. 290, January 29, 1977.

> "The 'protective' effect of smoking in patients at risk to DVT [deep vein thrombosis] clearly extends beyond those who suffer myocardial infarction."

Seltzer, C. C., "Stopping Smoking and CHD," The Lancet, p. 420, February 19, 1977.

> "The C.H.D. attack-rates of Framingham never smokers are substantially higher than for those who stopped smoking, and only slightly lower than those who continue to smoke cigarettes. This anomaly does not lend credence to the conclusion of the Framingham report on the effect on C.H.D. rates of stopping smoking. On the face of it, the figure would suggest that it is better to begin smoking and then to quit than never to have smoked at all. A more plausible explanation is that the various categories of smokers, quitters, and never smokers, are self-selected groups of men differing in socio biological and psychological characteristics leading to differential disease rates."

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

2026259314

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Ito, H. & D.M. Aviado, "Pulmonary Emphysema and Cigarette Smoke - Experimental Induction and Use of Bronchodilators in Rats," Archives of Environmental Health 16(6): 867-870, June, 1968.

> "The results are of particular interest because they question the validity of the widely accepted belief that cigarette smoke can promote the development of pulmonary emphysema."
>
> p. 865

Bauer, D.R., Medical World News 10: 34, August 22, 1969.

> "When Medical World News embarked on the current environmental health series. . .we wanted to photograph two lungs: one taken from a mature adult who had lived his life in the country and the other from an urban dweller. There was no difficulty in locating a predictable city lung blackened by years of inhaling polluted air. . . .
>
> "The search for a clean lung led south to Virginia and, finally, to Vermont. Both the lungs there were all discolored. Even amid the bucolic splendors of Vermont, reports Dr. Lawrence S. Harris, that state's chief medical examiner, it is increasingly difficult to find a clean lung in a mature adult. Only by photographing the lung of a four-month-old infant who died of epilepsy did we find the expected contrast."

Levine, E. R., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 1334-1339.

> "I believe that emphysema must be listed among the vascular diseases. As such, I cannot find any actual evidence that anything inhaled into the bronchial tree, cigarette smoke or anything else, has a causal relationship to the development of this disease."
>
> p. 1336

2026259315

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Roberts, K. E., "Emphysema and Enterobacterial Infection," Medical Counterpoint 2(3): 47, March, 1970.

> "In humans, only two lines of evidence have linked cigarette smoking and emphysema; one is statistical and the other is political. . . .The reasonable doubts of the minority are cultured by this study and require another trial in the court of facts before assigning emphysema to man's external rather than internal environment."

Rosenblatt, M. B., "Validity of Emphysema Mortality Data," Review of Allergy 24: 1019-1022, November, 1970.

> "Quantophrenia, the state of fanatic obsession with numbers, is a common affliction among epidemiologists often leading to unwarranted conclusions. . . . If our current breed of statisticians would take the time to study the history of emphysema during the past 300 years they would not be so zealous in implicating cigarettes as an etiologic agent."
>
> p. 1020

Aviado, D. M., et al., "Cigarette Smoke and Pulmonary Emphysema," Archives of Environmental Health 20: 483-487, April, 1970.

> "The experiments in rats have failed to support the widespread belief that cigarette smoke can induce experimental pulmonary emphysema."
>
> p. 487

2026259316

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Rosenblatt, M. B., "Emphysema: Historical Persective [sic]," Bulletin of N.Y. Academy of Medicine 48(6): 823-841, July, 1972.

> "It is difficult to reconcile the prevalence of emphysema in the 19th century with official statistics showing virtual nonexistence of the disease in the early decades of the 20th century. The present review is not concerned with the reasons for this disparity except to point out that the sudden apparent increase of emphysema in the United States may well be an artifact produced by revision of the International List of the Causes of Death in 1949 permitting emphysema, for the first time, to be accepted as a primary cause of death."
>
> p. 838

Dimond, J. L., "Chronic Bronchitis," New Zealand Medical Journal 75: 38, January, 1972.

> "Within the next decade English physicians will probably be agreeing with their Continental colleagues that chronic bronchitis is in actual fact a psycho-physiological disorder.
>
> ". . . although the rate of smoking per adult has increased threefold in the last 50 years there has been no increased incidence in standard mortality rates from bronchitis in males. . . ."

National Heart & Lung Institute, Submission, Hearings Before A Subcommittee of the Committee on Appropriations, House of Representatives, p. 292, March 28, 1973.

> "The cause or causes of emphysema are not known . . ."

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Sherman, J., et al., "A Health Research Group Study on Disease Among Workers in the Auto Industry," Washington, D.C.: Unpublished Paper Released September 7, 1973, p. 11.

> "These data . . . seriously challenge the traditional view by management and much of the medical profession that workers' lung and heart diseases are largely caused by cigarettes rather than by workplace poisons."

National Heart & Lung Institute, Submission, Hearings Before a Subcommittee of the Committee on Appropriations, House of Representatives, p. 281, March 26, 1974.

> "But we do not know the cause of pulmonary emphysema, how to stop its progress even if detected early, or how to prevent heart disease caused by emphysema."

Haydon-Baillie, M., FRCS, "Responsible Enthusiasm," World Medicine, London, Cutting #3096, June 19, 1974.

> "[F]irstly, I for one do not credit the simple belief that smoking is a cause of pulmonary disease; secondly, those persuaded to relinquish smoking tend to overeat thereafter; and thirdly, the review of statistics by Hinshaw and Garland shows inexplicable variations in the incidence of lung cancer in smokers, notes that cancer of the more affected larynx is comparatively rare and concludes that the lung cancer incidence in smoking females has been declining since 1960 and that in males it will decline from a peak in 1983."

2026259318

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Hammer, D. I., et al., "Air Pollution and Childhood Lower Respiratory Disease: Exposure to Sulfur Oxides and Particulate Matter in New York, 1972," Presented at the American Medical Association Air Pollution Medical Research Conference, San Francisco, California, pp. 1-8, December 5-6, 1974.

> "Reported lower respiratory disease morbidity was significantly higher among children exposed to ambient sulfur oxide and particulate air pollution than comparable children not so exposed."
>
> "[L]ower respiratory disease morbidity did not differ among children of smoking and nonsmoking parents."
>
> p. 6

Walker, W. J., "Headline Correction," Medical Tribune, p. 11, March 26, 1975.

> "The age-adjusted death rate from emphysema peaked in the United States in 1968 and has declined each year since then. . . . Instead of the fastest growing cause of death it turns out to be a 17% decline during a five year period!"

Ringler, Robert L., Testimony, Hearings of the Committee on Appropriations, House of Representatives, Volume 3, pp. 210-212, April 10, 1975.

> "The etiology, the cause, in other words, of the disease [emphysema] is really not known, to be truthful with you. It is thought to be associated with a number of factors, one of which may be a hereditary tendency to be more susceptible to it."
>
> p. 211
>
> "To a degree, I believe, it is hereditary and by that I mean an increased susceptibility to the disease."
>
> p. 212

2026259319

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Blot, W. J. & J. F. Fraumeni, "Arsenical Air Pollution and Lung Cancer," The Lancet, pp. 142-144, July 26, 1975.

> "The most likely explanation for the increased lung cancer mortality in this study is neighbourhood air pollution from industrial sources of inorganic arsenic. . . .
>
> "The carcinogenic effect of asbestos is not confined to workers, but may extend to surrounding neighbourhoods and families: our findings, although preliminary, suggest that the carcinogenic hazard of arsenic may cross plant boundaries into the general community.
>
> "It is possible that tobacco consumption is greater in counties with copper, lead, or zinc industries, but there is no reason to suspect this. The smoking habits of workers in copper smelters were not unusual."
>
> p. 144

Brown, S. M., et al., "Effect on Mortality of the 1974 Fuel Crisis," Nature 257: 306-307, September 25, 1975.

> "The 1974 fuel crisis was a natural experiment. It presented the opportunity to test the hypothesis that a decrease in vehicular exhaust fumes would have a beneficial effect on health. . . . Dramatic decreases were noted in death rates for several major categories of disease. . . .The disease showing the greatest relative change was chronic lung disease."
>
> p. 306

Newhouse, M., et al., "Lung Defense Mechanisms," New England Journal of Medicine 295(18): 990-998, October 28, 1976.

> "Regardless of the exact mechanism of tissue destruction associated with deficiency of alpha antitrypsin, it is clear that lack of this factor may allow the inflammatory process to proceed unchecked and may be the direct causative factor responsible for the pathogenesis of continued lung destruction leading to emphysema."
>
> pp. 993-994

2026259320

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Kornegay, Horace R., President, The Tobacco Institute, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, February 19, 1976.

> "[S]cientists do not understand the pathogenesis of bronchitis or emphysema (any more than they do the pathogenesis of cancer or cardiovascular disease). Scientists do generally agree that chronic bronchitis and emphysema are multifactorial. But which of the observed factors, if any, plays a role in their causation remains unknown."
>
> p. 360

2026259321

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Sterling, Theodor D., "Does Smoking Kill Workers or Working Kill Smokers? or The Mutual Relationship Between Smoking, Occupation and Respiratory Disease," Presentation for School of Workers, University of Wisconsin Extension, pp. 1-17, April 18-22, 1977.

> "While cigarette smoking has been widely accepted by physicians and public health workers alike as the major cause of many lung diseases, a score of scientists have continued to point to the flaws, irregularities, and contradictions in the smoking and health data. Some even feel that some of the effects of occupational exposures may have been MASKED by poorly designed and ineptly analyzed smoking studies. Enough evidence exists now to seriously question the widespread conviction that cigarette smoking is the primary cause of lung disease, especially cancer and chronic obstructive disease."
>
> p. 1

> "[I]n our recent study of pulp and paper mill workers with severe respiratory deficiencies, we found that 62 percent had a history of smoking while such history was found among 66 percent of the other workers. Thus smoking was less prevalent among workers with signs of respiratory failure than among those with no such signs. (But there was a relation to high exposure to fumes.) In another survey of about 1800 compensation claims we found that 56 percent of disabled workers had a smoking history which is low for blue collar workers.
>
> "In analyzing the available evidence, we do not find support for claims that smoking is the major hazard to workers' lungs - it's their jobs which seem to cause their illness."
>
> p. 6

2026259322

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Sterling, Theodor D., "Does Smoking Kill Workers or Working Kill Smokers? or The Mutual Relationship Between Smoking, Occupation and Respiratory Disease," Presentation for School of Workers, University of Wisconsin Extension, pp. 1-17, April 18-22, 1977.

> "The ease with which lung cancer can be produced in animals using a wide variety of industrial pollutants and the failure so far to produce lung cancer by tobacco smoke are important factors that have long been neglected in trying to assess the effect of smoking on lung cancer and other lung diseases."
>
> p. 12

Huber, G. L., "The Effect of Chronic Exposure to Tobacco Smoke on the Antibacterial Defenses of the Lung," Bulletin Europeen de Physiopathologie Respiratoire 13(1): 145-56, 1977.

> "[M]any alterations in the metabolism of alveolar macrophages are caused by tobacco but as is evident from the data presented these alterations have not impaired function of the cell and may, indeed, have activated the cell."
>
> p. 152

> "We would conclude, on the basis of our multidisciplinary approach to the effects of experimental tobacco smoke exposure on the defense system of the lung, that pulmonary antibacterial activity does not seem to be impaired by cigarette smoke under our experimental conditions. The contradictory implications of others may be based, for the most part, on data obtained from unrealistically high smoke exposure levels, and bear little relevance to the potential effects of cigarette consumption by man. Our studies to date on the metabolism and function of alveolar macrophages harvested from smoke-exposed animals indicate that the cell is probably activated, not impaired, and has a normal or enhanced phagocytic capacity."
>
> p. 153

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

2026259324

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

CANCER

Hirayama, T., "Epidemiology of Stomach Cancer," Cancer Research 11: 3-19, 1971.

> "On the other hand the frequency of milk intake was significantly lower in the stomach cancer group compared with the controls. . . .No significant difference was observed with regard to the frequency of intake of other items. . . . No striking association was observed with the habits of smoking and drinking. From these results it is apparent that excessive intake of highly salted foods and the lesser intake of milk are closely associated with the occurrence of stomach cancer."
>
> p. 13

ULCER

Debas, H. T. & M. M. Cohen, "Effect of Smoking on Gastric Secretion Stimulated by Pentogastrin," The Lancet: 43-44, January 1, 1972.

> "We remain convinced that there is no evidence that either smoking or nicotine causes any significant alteration to the gastric secretory response of normal humans and that the effect of these agents on peptic-ulcer patients has not yet been adequately tested."
>
> p. 44

2026259325

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

CANCER

Stell, P. M., "Smoking and Laryngeal Cancer," The Lancet, pp. 617-618, March 18, 1972.

> ". . .the incidence of laryngeal cancer has remained more or less constant for 70 years--a period in which tobacco consumption and the incidence of cancer of the lung have risen sharply. . . .If cigarette smoke is carcinogenic it seems reasonable to suppose that it should predispose to cancer of the mouth, pharynx, and larynx--but there is no irrefutable evidence that it does so."
>
> p. 617

> "Any data showing a correlation between heavy cigarette smoking and laryngeal carcinoma must be interpreted with caution because in the entire population the incidence of laryngeal carcinoma has been remarkably constant; this is even more striking when the incidence of laryngeal carcinoma is compared to the incidence of lung cancer, and to the annual consumption of tobacco and of cigarettes."
>
> p. 618

ULCER

Cooper, P. & S. H. Tolins, "Relationship Between Smoking History and Complications Immediately Following Surgery for Duodenal Ulcer," The Mount Sinai Journal of Medicine 39(3): 287-293, May-June, 1972.

> "A cooperative study was conducted by the Veterans Administration with the express purpose of comparing four surgical procedures as a treatment regimen in patients with duodenal ulcer disease. . . . The results give no evidence of an association between smoking and complications, nor between smoking and operative mortality, nor between smoking and days to discharge. The most obvious trend is probably predictable, namely patients over 50 have more complications and a higher mortality rate than those under 50."
>
> p. 291

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

THROMBOSIS

Handley, A. J. & D. Teather, "Influence of Smoking on Deep Vein Thrombosis After Myocardial Infarction," British Medical Journal 3: 230-231, July 27, 1974.

> "As part of a study of the factors affecting the risk of deep vein thrombosis after myocardial infarction a surprising and unexplained finding was that non-smokers had a significantly higher incidence of thrombosis than cigarette smokers."
>
> p. 230

THROMBOSIS

Marks, P. & Emerson, P. A., "Increased Incidence of Deep Vein Thrombosis After Myocardial Infarction in Non-Smokers," British Medical Journal, pp. 232-234, July 27, 1974.

> "The incidence of deep vein thrombosis became 52% in the non-smokers and only 5.4% in the smokers. This difference is again highly significant.
>
> "There was no significant difference in the incidence of deep vein thrombosis between those who smoked a pipe and those who smoked cigarettes (none smoked both). Furthermore there was no positive correlation between the numbers of cigarettes smoked and the incidence of deep venous thrombosis.
>
> "The overall frequency of venous thrombosis after myocardial infarction in this series was similar to that reported by other workers, but in our patients the non-smokers were much more likely to develop deep vein thrombosis after their myocardial infarctions than were the smokers."
>
> p. 233

2026259327

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

STROKE

Nomura, A., et al., "Cigarette Smoking and Strokes," Stroke 5(4): 483-486, July-August, 1974.

> "If the relationship between cigarette smoking and atherosclerotic strokes is not age dependent, it follows that cigarette smoking may not be associated with the atherosclerotic process per se."
>
> p. 486

ALLERGY

Taylor, G., "Tobacco Smoke Allergy - Does It Exist," Chapter 3.5 in Environmental Tobacco Smoke Effects on the Nonsmoker (R. Rylander, Ed.) University of Geneva, pp. 50-55, 1974.

> "The question of correlation between positive skin tests to tobacco extracts and clinical response to tobacco smoke presents great difficulties. . . .The finding that 12% of normal smokers have positive skin responses to tobacco extract yet continue to smoke, would cast considerable doubt on the relationship of positive weal and flare skin response to the clinical response of the subject to tobacco smoke exposure."
>
> p. 52

GLAUCOMA & OCULAR HYPERTENSION

Morgan, R. W. & S. M. Drance, "Chronic Open-Angle Glaucoma and Ocular Hypertension - An Epidemiological Study," British Journal of Ophthalmology 59: 211-215, August 15, 1975.

> "Analysis demonstrated no association between smoking and disease; smoking histories of cases and controls were virtually identical. The effect of smoking on the vascular component of chronic simple glaucoma appears minimal, if present at all."
>
> p. 213

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

SEXUAL FUNCTION & FERTILITY

Sterling, T. D., "A Critical Review of Reports on the Effect of Smoking on Sex and Fertility," The Journal of Sex Research 11(3): 201-217, August, 1975.

> "In general, it is unwarranted to conclude that smoking interferes with sexual activity or fertility."
>
> p. 216

CANCER

Burch, P. R. J., Problems in the Interpretation of Cancer Statistics with Special Reference to Lung Cancer," Journal Society of Occupational Medicine 25: 2-10, 1975.

> "My analysis. . .indicates that most human cancers, as recorded in national mortality statistics, are spontaneous in origin. I infer that they arise, in genetically predisposed persons, as the result of the intrinsic instability of genes in stem cells of the central system of growth-control."
>
> p. 9

BLOOD PRESSURE

Hrustic, O. & M. Saric, "Odnos Navike Pusenja I Uzivanja Alkohola Prema Visini Arterijskog Krvnoh Tlaka U Skupini Aktivnog Stanovnistva," (Study of smoking habit and alcohol consumption in relation to arterial blood pressure in a group of workers) Arhiv Za Higijenu Rada I Toksikologiju 26(1): 15-22, 1975.

> "In a group of 759 male industrial workers aged 20-59 (mean age 37 years) the arterial blood pressure was studied in relation to smoking habit and alcohol consumption.
>
> "The results indicate that there is no association between smoking habit and higher values of arterial blood pressure. On the average, nonsmokers even had a somewhat higher blood pressure than smokers."
>
> p. 22

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

BRONCHITIS

Smith, B., "Cancer Incidence," New Scientist, pp. 304-305, February 5, 1976.

> "There is a current practice by the anti-smoking lobby, of taking a multifactorial condition such as coronary occlusion or bronchitis, in which smoking may be one factor, and citing it as the primary cause. Figures currently given by Dr. Owen [U.K. Minister of Health] of the annual mortality due to smoking must use this technique. They are too large to be real. After all, half the adult population does not smoke."
>
> p. 305

MIGRAINE

Volans, G. N. & C. M. Castleden, "The Relationship Between Smoking and Migraine," Postgraduate Medical Journal 52(604): 80-82, February, 1976.

> "From this study it is concluded that the theory of an association between smoking and migraine has not been proved and that no evidence was found to suggest that active or passive smoking is a factor in the aetiology of migraine."
>
> p. 82

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

ALLERGY

Aviado, D. M., Statement Before the Public Hearing Before the Council of the District of Columbia Committee on Transportation and Environmental Affairs, pp. 1-6, April 27, 1976.

> "Reported irritation of the eyes and nose has been explained as a nonallergic response because the reaction occurred in both allergic and nonallergic individuals. True tobacco smoke allergy is believed to be quite rare. . . The method of determining whether an allergy exists has not been settled although many allergists make use of a skin test using tobacco leaf extract. Such skin testing is not at all comparable to exposure to tobacco smoke. There are, of course, many substances (such as pollens and household dusts) which may elicit an allergic response. It seems impratical [sic], however, to seek to protect allergic individuals by regulating one out of countless sources of allergens."
>
> p. 2

ALLERGY

McDougall, J. C. & G. J. Gleich, "The Tobacco Allergy - Fact or Fancy," Presented at the American Academy of Allergy Meeting, San Juan, Puerto Rico, June, 1976.

> "[W]e can say that there is a protein in tobacco which is antigenic in rabbits. We were unable to detect any humoral immune response to this protein or to tobacco smoke or smoke treated human serum albumin with the serum of bronchial secretions of human subjects. We did find that some subjects had positive skin test responses to tobacco protein but that this does not correlate with the presence or absence of symptoms. We were also unable to demonstrate any specific IgE binding to tobacco protein or smoke treated human serum albumin in the serum of patients who reported allergic symptoms to tobacco or tobacco smoke."
>
> p. 5

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

ADDICTION

Aviado, Domingo M., Professor of Pharmacology, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "[T]here seems to be a growing popular belief that smoking is literally addictive. Even the 1964 Advisory Committee's report to the Surgeon General concluded that this was not correct. Further, from my review of the literature, it is apparent neither nicotine nor tobacco smoke should properly be considered addictive."
>
> p. 30

ALLERGY

Justus, David E. & David A. Adams, "Evaluation of Tobacco Hypersensitivity Responses in the Mouse," Int. Arches Allergy app. Immun. 51: 687-695, 1976.

> "Although it was possible to obtain good sensitization in animals by immunization with tobacco leaf extract, similar attempts using either water-soluble tobacco smoke condensates . . . or prolonged exposure of mice to whole smoke (done by animal smoking apparatus) were totally unsuccessful."
>
> pp. 693-694

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

CANCER

Sterling, Theodor D., "Does Smoking Kill Workers or Working Kill Smokers? or The Mutual Relationship Between Smoking, Occupation and Respiratory Disease," Presentation for School of Workers, University of Wisconsin Extension, pp. 1-17, April 18-22, 1977.

> "The expected proportion of all cancer deaths among males is approximately 17 per cent indicating that the cancer rate among asbestos workers is about three times that of comparable U.S. males. Recently Johns Manville insisted on writing a no-smoking clause in a union contract. Perhaps this is just one more attempt to divert attention away from the hazard of work with asbestos."
>
> p. 6

CANCER

Sterling, Theodor D., "Does Smoking Kill Workers or Working Kill Smokers? or The Mutual Relationship Between Smoking, Occupation and Respiratory Disease," Presentation for School of Workers, University of Wisconsin Extension, pp. 1-17, April 18-22, 1977.

> "Smoking appears to have been used to divert attention away from the effects of occupational and of environmental exposures. . . . It is of the greatest importance in this connection that to our knowledge not one Cancer Registry in Canada and the U.S. bothers to determine the occupation of lung or other cancer cases although information on smoking always is included."
>
> p. 14

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Bair, W.J., et al., "Apparatus for Direct Inhalation of Cigarette Smoke by Dogs," Journal of Applied Physiology 26(6): 847-850, June, 1969.

> "Most of these experiments, such as exposing animals to cigarette smoke in a smoke chamber or introducing cigarette smoke to the anesthetized animal through a tracheotomy aperture, lack similarity to human smoking habits. The validity of extrapolating results from such experiments to possible effects in man is therefore highly questionable."
>
> p. 847

Buhler, V.B., Letter to Hon. Tim Lee Carter, Congressional Record, E2639, March 26, 1970.

> ". . . the unpublished report by Dr. Auerbach must be viewed with considerable restraint. I sincerely hope that his reporting of 'early squamous cell bronchial carcinoma' in only two dogs will not be viewed by your Committee as scientific proof as to whether or not cigarette smoking has been established as the cause of lung cancer in humans."

Buhler, V.B., Letter to Hon. Tim Lee Carter, Congressional Record, E1856, March 15, 1971.

> "My previous concern about this study has been confirmed by the finally published article, which so completely fails to bear out the claims announced at the American Cancer Society's press conference last February."

Sterling, T.D., "Comment on Smoking Dogs," Archives of Environmental Health 22: 631-32, May, 1971.

> "The need for providing adequate controls, especially in experiments that involve surgical incision and bypassing of natural functions, is so deeply engrained in biological science that its violation in this important piece of work defies comprehension. Indeed, the consequences of this work will be confusion rather than enlightenment."
>
> pp. 631-32

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Hickey, R., "Environmental Chemical Mutagens: Are They Health Hazards?" (Excerpt printed in Ecolibrium) Ecolibrium 1(1): 12, June, 1972.

> "It is a much too common part of scientific methodology these days, in dose-response experiments in biology, to employ unrealistically high doses . . . of some chemical at varying levels in studies on small numbers of inbred experimental animals. It is also commonplace to extrapolate high level findings into quite low dosage regions in which it is quite difficult to detect effects, and to assert on the basis of subjective judgment or opinion something about the effects in this low dosage region and what it means to human health. How much regulation or law has been made by such speculation I will leave to you to estimate. Based on my understanding of science and scientific methods, extrapolation into unknown regions is scientifically invalid, and should be called by its proper name: speculation."

Hirth, R. S. and G. H. Hottendorf, "Lesions Produced by a New Lungworm in Beagle Dogs," Veterinary Pathology 10: 385-407, 1973.

> "Canine pulmonary lesions that were reported to be neoplastic and experimentally produced are similar to some of the changes present in our lungworm-infected dogs. . . . [I]t is likely that this new canine lungworm is present in many kennels throughout the United States that raise Beagles for research."
>
> pp. 405-406

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag, Stockholm, pp. 341-362, 1973.

> "The animal models used to study the development of arteriosclerosis, and in particular coronary arteriosclerosis, ischaemic heart disease and myocardial infarction are not representative of the human disease. Consequently, though much work has been invested in these animal experiments few conclusions can be drawn from the results of these to the clinical or human situation. This is especially true for the huge literature on dietary studies using maximally distorted diets with nonrealistic relations when compared with ordinary clinical situations. This is also true for animal studies on behaviour, cigarette smoking (or influence of nicotine or of carbon monoxide).
>
> p. 350

Anonymous, "Animal Study Shows Intriguing Link Between Chronic Stress, Cancer," Journal of the American Medical Association 233(7): 757-758, August 18, 1975.

> "[According to Dr. Riley, Chairman of the Department of Microbiology at the Pacific Northwest Research Foundation in Seattle] 'Specific studies in our laboratory have demonstrated the adverse physiological and biochemical consequences of even the mildest handling and experimental manipulation of mice.'" .
>
> p. 757

Gifford-Jones, W., "'Cancerphobia': The Universal Disease," Chapter 11 in The Doctor Game, Mc Clelland & Stewart, Ltd. (Toronto, 1975), pp. 131-138.

> "Backers of the smoking-lung cancer theory frequently refer to experiments in which skin cancers are produced in mice by repeatedly painting their skins with tar. But medical journals have severely criticized these experiments. The quantity of tar needed to produce the cancer is fantastically higher than that found in cigarette smoke. To expose yourself to the same amount of tar, you would have to smoke 100,000 cigarettes a day!"
>
> p. 136

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Hickey, R. J., et al., "Cigarette Smoke As A Carcinogen?", American Review of Respiratory Disease 111: 105-106, 1975.

> "In studies in which experimental animals were exposed to realistic levels of tobacco smoke, the results have generally failed to support the hypothesis that smoking causes lung cancer."
>
> p. 105

Georgi, Jay R., "Filaroides hirthi: Experimental Transmission Among Beagle Dogs Through Ingestion of First-Stage Larvae," Science 194(4266): 735, November 12, 1976.

> "Infection with F. hirthi [lungworms] is thus widespread in beagle dogs used for research and causes lesions that interfere with the pathological evaluation of experimental material."
>
> p. 735

Sommers, Sheldon C., M.D., Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, February 19, 1976.

> "Certain false alarms like the Auerbach beagle dogs, and other studies of rats and mice, have stirred hope that a model had been achieved, but no cancers that grew, spread, and led to death like human squamous cell lung carcinomas have been reported in animals.
>
> "After 45 years of inhalation research, and although expensive efforts continue, no success has been achieved in producing experimental lung cancers in any reasonable, or even small numbers of experimental animals."
>
> p. 272

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Wehner, Alfred P., et al., "Increased Life Span and Decreased Weight in Hamsters Exposed to Cigarette Smoke," Archives of Environmental Health 31: 146-153, 1976.

> "In this paper, the effect of chronic exposure to cigarette smoke on life span and body weight [of hamsters] is described."
>
> p. 146

> "The most surprising result of our experiments [with hamsters] was the fact that groups having received chronic exposure to cigarette smoke lived significantly. . .longer than their sham-smoke-exposed cohorts and the controls."
>
> p. 151

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Brower, L.P., "Smoking & Cancer," The New York Times, February 15, 1970.

> "All this experiment proves is: Whether or not smoking causes lung cancer in beagles is as inconclusive now as it was before the experiment was carried out."

Editorial, "The Cigarette-Cancer Dispute," The New York Times, May 9, 1970.

> "Scientific truth is determined ultimately by open examination of data and full discussion by all competent persons in a field. The Hammond-Auerbach results should be scrutinized intensively by all interested parties."

Editorial, Environment 15(4): 21, May, 1973.

> "The report was first rejected by the Journal of the American Medical Association, and on at least two subsequent occasions, independent pathologists have reviewed Dr. Auerbach's records and found no evidence of cancer.
>
> "Much of this research, like that of Dr. Auerbach, has been supported by the American Cancer Society, which has so far refused to allow examination of the raw data produced in its extensive epidemiological studies."

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Kensler, C. J., "Components of Pharmacologic Interest in Tobacco Smoke," Ann. N. Y. Acad. Sci. 90(1): 43-47, 1960.

> "In conclusion, it should be pointed out that estimation of pharmacological importance of the amounts of the compounds present in tobacco smoke is best related to the response of the average individual, and should not be generalized to include individuals who are extremely sensitive because of genetic or other reasons, or to those individuals who, as a result of differential aging or the presence or history of disease, may differ markedly from the average or normal in their response."
>
> pp. 46-47

Bentley, H. R. & J. G. Burgan, "Cigarette Smoke Condensate: Preparation and Routine Laboratory Estimation," Tob. Man. Stand. Comm. (4): 2-9, 1961.

> "The main conclusions to be drawn from the above experimental results are as follows. . .The same brand of cigarettes made at different times can have different yields of smoke condensate. The yield of smoke condensate from a cigarette varies with the conditions under which the cigarette is smoked."
>
> p. 8

Davies, R. F. & J. K. Whitehead, "A Study of the Effects of Altering the Tar/Nicotine Ratio in Experimental Tobacco Carcinogenesis," British Journal Cancer 24(1): 191-194, March, 1970.

> "There was no statistically significant difference in specific mouse skin carcinogenicity between smoke condensate from plain, flue-cured tobacco cigarettes with a normal tar to nicotine ratio and condensate from filter-tip cigarettes made from selected flue-cured tobaccos with a reduced tar to nicotine ratio."
>
> p. 191

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Okun, R., Testimony, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U. S. Senate, February 1, 3 & 10, 1972, pp. 230-234.

" . . . a recent study has failed to show any statistically significant differences in animal effects between tar from one type of cigarette with a normal tar-nicotine content and the tar from filter-tipped cigarettes made from other types of tobacco with reduced tar-nicotine."
p. 230

"From my experience and review of the scientific literature and the recent Surgeon General's report, I cannot tell the significance of nicotine content, tar content, and potential carbon monoxide levels derived from cigarette smoke on health.

"However, a false sense of significance, if not security, can be derived by the nonscientific public from government establishment or approval of maximum levels of these constituents."
p. 232

"There are many things that bear on tar content of a cigarette that have to do with more than just the grams of tobacco that exist in a cigarette:

> "The type of tobacco, where the tobacco is grown on the plant and whether sheets are used, stems are used, et cetera, or whether it is chemically treated.

"All these things bear on the quality of tar. You may find that the weight of tar between a cigarette is the same, but qualitatively quite different."
pp. 233-234

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Soloff, L. A., Statement, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U.S. Senate, February 1, 3, & 10, 1972 (as quoted by Senator Marlow Cook, Closing Statement, p. 286).

> "In conclusion, the data linking heart disease and smoking are still questionable. Studies with particular reference to twins in the United States and Sweden and epidemiological studies in many countries fail to show a correlation between smoking and coronary heart disease. Human studies on smoking that utilize highly artificial circumstances are of questionable validity. Such data do not provide support for the proposed arbitrary limitation on so-called 'incriminated agents' in cigarette smoke."

Rigdon, R. H., Statement, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U.S. Senate, February 1, 3, & 10, 1972 (as quoted by Senator Marlow Cook, Closing Statement, p. 286).

> ". . .to my knowledge, it has never been scientifically established that tobacco smoke condensate is harmful or hazardous to smokers.
>
> ". . . my experiments emphasize the fact that much additional information is needed before we draw any conclusions about the effects of tobacco smoke condensate or any of its constituents on smokers."

Furst, A., Testimony, Hearings before the Consumer Subcommittee of the Committee of Commerce, U.S. Senate, February 1, 3, & 10, 1972, pp. 203-217.

> ". . .setting limits on compounds formed in tobacco smoke or condensate would be to act in what is nearly a scientific vacuum as far as good experimental data is concerned."
>
> p. 207
>
> "Condensates will vary depending on the conditions of burning and on the composition of cigarettes; unless you absolutely specify every single factor, moisture content, tobacco content, burning zone, rate of burning, you will get different condensates."
>
> p. 215

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag (Stockholm 1973) pp. 341-362.

> "The animal models used to study the development of arteriosclerosis, and in particular coronary arteriosclerosis, ischaemic heart disease and myocardial infarction are not representative of the human disease. Consequently, though much work has been invested in these animal experiments few conclusions can be drawn from the results of these to the clinical or human situation. This is especially true for the huge literature on dietary studies using maximally distorted diets with fats, carbohydrates, proteins and vitamins in nonrealistic relations when compared with ordinary clinical situations. This is also true for animal studies on behaviour, cigarette smoking (or influence of nicotine or of carbon monoxide)."
>
> p. 350

Schechter, Martin D., "Effect of Nicotine on Response to Frustrative Non-Reward in the Rat," European Journal of Pharmacology 29: 312-315, 1974.

> "If smoking lowers hostility in humans, as indicated by recent investigations, . . . this may be a factor in the continuance of the smoking habit, and may be viewed as a 'beneficial' effect for a much-maligned habit."
>
> p. 314

Eskwith, I.S., "Etiology of Atherosclerotic Heart Disease," American Heart Journal 90(6): 809-810, December, 1975.

> "So far no one has found any direct effect of nicotine on the heart other than it somewhat increases cardiac output. Cardiologists today are sending poor middle-aged men out on the road, exercising, to achieve therefore, the same results that could be obtained by a half a package of cigarettes."
>
> p. 809

2026259345

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Lloyd, J. P. F., "Segregated Smokers," British Medical Journal 1(6006): 398, February 14, 1976.

> "Surely the minute traces of tobacco tars joining those of many other 'indictable substances' in the atmosphere can be of little significance."

Master, Kenneth, "Carbon Monoxide, Atherosclerosis, and Natives of the Highlands of New Guinea," New England Journal of Medicine 294(10): 557, March 4, 1976.

> "[E]xposure to elevated levels of carbon monoxide does not induce arteriosclerotic vascular disease in natives of the highlands of New Guinea. It is likely that inhalation of carbon monoxide alone does not directly cause atherosclerosis in man but, in combination with dietary, stress, hereditary, and other unknown factors, may predispose to it."

Forbes, W. F., et al., "Studies on the Nicotine Exposure of Individual Smokers. I. Changes in Mouth-Level Exposure to Nicotine on Switching to Lower Nicotine Cigarettes," The International Journal of the Addictions 11(6): 933-950, 1976.

> "[M]ost subjects reduced their [nicotine] exposure while smoking the lower nicotine brand, and displayed little tendency to increase their relative daily mouth-level exposure compared to a smoking machine while smoking the lower nicotine brand."
>
> p. 949

2026259346

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Okun, Ronald, Associate Professor of Medicine and Medical Pharmacology and Therapeutics, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "Accordingly, as I stated in 1972 and continue to believe, I cannot tell the significance of nicotine or 'tar' derived from cigarette smoke on human health and certainly there is no scientific basis for the claim that 'tar' and nicotine have been shown to be harmful to human smokers."
>
> pp. 155-156

Seltzer, Carl C., Senior Research Associate, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "[I]t has never been argued by the proponents of the cause-effect theory of smoking and heart disease that 'tar' has a harmful effect on CHD. Nicotine has been suggested, but it should be pointed out that after the Surgeon General's Advisory Committee on Smoking and Health in 1964 had reviewed all the evidence, rejected the possible role of nicotine with the statement that 'No additional or unique cardiovascular effects [nicotine] have been demonstrated. . .to account for the observed association of cigarette smoking with an increased incidence of coronary disease."
>
> p. 79

Hine, Charles H., Clinical Professor of Pharmacology and Preventive Medicine, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "[Cardiovascular responses to nicotine] are of limited duration in time and to the extent that they can be activated by the nicotine in cigarette smoke, last only for a moment."
>
> p. 135

2026259347

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Hine, Charles H., Clinical Professor of Pharmacology and Preventive Medicine, Statement, Hearings Before the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, 1976.

> "We simply do not know whether cigarette 'tar' has any relationship to human disease."
> p. 139

Weber, Karl H., "Recent Changes in Tobacco Products and Their Acceptance by the Consumer," Paper Presented at the 6th International Tobacco Scientific Congress, Tokyo, pp. 15-80, 1976.

> "The lower ['tar' and nicotine] yield of 'lighter' cigarettes is, . . . not compensated by an increase in cigarette consumption."
> p. 21

2026259348

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Anonymous, "Disagrees On Pilot Smoking," Aberdeen AM News, Aberdeen, South Dakota, 4/29/76.

> "Psychology Prof. Dr. Norman Heimstra, University of South Dakota (USD), . . . dean of the USD graduate school and director of the Human Factors Laboratory at USD, says those claims . . . that smoking inhibits performance, especially when carbon monoxide combines with high altitudes . . . are not documented.
>
> "'If anything, in some situations smokers may make the better pilots,' he said in a prepared statement."

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Fifer, W. R., "Air Pollution - A Primer For Practicing Physicians," Bulletin of the St. Louis Park Medical Center 13(3): 1-7, 1969.

> "There is mounting evidence that the serious chronic lung diseases such as lung cancer, chronic bronchitis and emphysema may result, at least in part, from air pollution."
>
> p. 6

McCall, M.G. & N.S. Stenhouse, "Deaths From Lung Cancer in Australia," The Medical Journal of Australia, pp. 524-525, March 6, 1971.

> "The effect of an environmental agent such as air pollution would be strongly supported by evidence that the death rate from lung cancer in British immigrants increases with increasing periods of residence in England before immigration to Australia. . . .Since smoking habits do not vary greatly between the countries studied, the findings reported here strongly support Dean's conclusion that the role of air pollution in the genesis of lung cancer has been seriously underestimated."
>
> p. 525

Eisenbud, M., & L. R. Ehrlich, "Carbon Monoxide Concentration Trends in Urban Atmospheres," Science, pp. 193-194, April 14, 1972.

> "The predominant modern source of CO is the exhaust gases from the internal-combustion engine. Approximately 97 percent of the CO in the air of New York City is currently attributable to this source."
>
> p. 193

2026259351

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Lave, B. & E. P. Seskin, "Air Pollution, Climate, and Home Heating: Their Effects on U. S. Mortality Rates," American Journal of Public Health 62: 909-916, July, 1972.

> "These studies make it apparent that there is a close association between mortality rates and air pollution. This investigation strengthens the conclusions cited in a previous work that mortality rates could be lowered substantially by abating air pollution."
>
> p. 915

"Work in America", Report of a Special Task Force to the Secretary of Health, Education and Welfare, December, 1972.

> "In addition to heart disease, several other illnesses have been found to be highly associated with occupational stress. The most convincing evidence pertains to peptic ulcers, and both arthritis and rheumatoid arthritis. Links also have been suggested for stroke and gout (a form of arthritis)."
>
> p. 66

Sterling, T. D., "Difficulties of Measuring the Effects of Air Pollution vs. Effects of Smoking," Paper Presented Before the American Association for the Advancement of Science, Washington, D. C., December 27, 1972.

> "A study of the air pollution and smoking effects inevitably then reaches two conclusions:
> 1. At a technical level, existing evidence is derived from inadequate studies. . . .
> 2. A pall of secrecy hangs over much of the crucial information that is needed to form valid conclusions about health effects either of smoking or of occupational and community pollution."
>
> pp. 18-19

2026259352

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Hueper, W. C., "Medicolegal Considerations of Occupational and Nonoccupational Environmental Cancer," Chapter 38 (VII) in: Lawyers Medical Cyclopedia of Personal Injuries and Allied Specialties (Revised Vol. 5, Part B), Frankel, C.J. (Editor), The Allen Smith Co., (Indianapolis, 1972), pp. 301-302.

> "Occupational carcinogenic agents encountered during regular occupational activities furnish the majority of known environmental carcinogens. . . .While many millions of workers, probably even the majority, have some contact with one or several of such agents, carcinogenic occupational hazards and the occurrence of occupational cancers have been established by epidemiologic studies for only a comparatively small fraction of the exposed working population, because neither industry nor governmental public health agencies have been interested sufficiently to determine the presence and scope of cancer hazards among the bulk of workers exposed."

Hueper, Wilhelm C., "Lung Cancer and Smoking in Perspective," Lawyers' Medical Cyclopedia....of Personal Injuries and Allied Specialties, Charles J. Frankel, Ed., Revised Vol. Five, The Allen Smith Company, Indianapolis, pp. 559-568, 1972.

> "Human exposure to many of these agents [potential human carcinogens] is not only widespread, but also often intense, and most of them are used without observing any real precautions. This lack of precautions may continue as long as the exposed public can be persuaded that the main lung cancer hazards are limited to cigarette smoking."
>
> p. 561

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Sterling, T.D., "Air Pollution and Smoking," Environment 15(6): 3-5, 25-26, July/August, 1973.

"However in the enthusiasm of many of our colleagues in attacking cigarette smoking as the major public health problem, the effect of cigarette smoking has been increasingly contrasted with that of pollutants and industrial exposure until today a real question exists if cigarette smoking is not diverting attention from the effects of occupational exposure on industrial workers and of the increasing air pollution burden on the population of our communities."

p. 3

"The juxtaposition between smoking and the effects of industrial and community pollution are of considerable importance and need to be sanely reassessed. There is also the possibility that the fervor with which many environmentalists attack smoking will be used to protect present uses of organic fuels from pressures exerted on them by air pollution research findings."

p. 4

"To begin with, many specific lung diseases are known to be caused by particles in the industrial environment. . . .While the effects of these chemicals, working by themselves or together, are largely unexplored, it has been shown that many substances in the industrial and community environment that exist in significant quantities possess considerable carcinogenic potential."

p. 5

"Many studies have concluded outright that the major effect on pulmonary disease and lung cancer appears to be carried by environmental pollution and not by smoking."

p. 25

2026259354

Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Sherman, J., et al., "A Health Research Group Study on Disease Among Workers in Auto Industry," Washington, D.C.: Unpublished Paper Released September 7, 1973, p. 11.

> "These data. . .seriously challenge the traditional view by management and much of the medical profession that workers' lung and heart diseases are largely caused by cigarettes rather than by workplace poisons."

Stewart, R.D., et al., "Carboxyhemoglobin Concentrations in Blood From Donors in Chicago, Milwaukee, New York, and Los Angeles," Science 182: 1362-1364, December 28, 1973.

> "Among the nonsmokers, 76 percent in Los Angeles, 74 percent in Chicago, 35 percent in New York, and 26 percent in Milwaukee had COHb saturations in excess of 1.5 percent, indicating CO exposure in excess of that permitted by air quality standards."
>
> p. 1363

Hammer, D. I., et al., "Air Pollution and Childhood Lower Respiratory Disease: Exposure to Sulfur Oxides and Particulate Matter in New York, 1972," Presented at the American Medical Association Air Pollution Medical Research Conference, San Francisco, California, pp. 1-8, December 5-6, 1974.

> "Reported lower respiratory disease morbidity was significantly higher among children exposed to ambient sulfur oxide and particulate air pollution than comparable children not so exposed."
>
> "[L]ower respiratory disease morbidity did not differ among children of smoking and nonsmoking parents."
>
> p. 6

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Mancuso, T. F. & T. D. Sterling, "Lung Cancer Among Black and White Migrants in the U.S.," Journal of the National Medical Association 67(2): 106-112, March, 1975.

> "Existing evidence does not support the smoking hypothesis. . . .In fact, only about half as many blacks smoked more than one pack a day as did whites (both in the North and South). . . .We think it reasonable to assume that smoking is completely unrelated to the difference in lung cancer between blacks and whites and migrants and non-migrants."
>
> p. 108

> "Implicated are some factors associated with place of birth in the U.S. and in particular in the South and the influences of early years of life and exposure to adverse industrial environments subsequent to migration. . . .Our findings support the hypothesis that lung cancer, at least among the blacks, may be largely a result of migration and occupational exposure to chemical dusts and fumes in the industrial employment."
>
> p. 111

Blot, W. J. & J. F. Fraumeni, "Arsenical Air Pollution and Lung Cancer," The Lancet, pp. 142-144, July 26, 1975.

> "The most likely explanation for the increased lung-cancer mortality in this study is neighbourhood air pollution from industrial sources of inorganic arsenic. . . .
>
> "The carcinogenic effect of asbestos is not confined to workers, but may extend to surrounding neighbourhoods and families: our findings, although preliminary, suggest that the carcinogenic hazard of arsenic may cross plant boundaries into the general community.
>
> "It is possible that tobacco consumption is greater in counties with copper, lead, or zinc industries, but there is no reason to suspect this. The smoking habits of workers in copper smelters were not unusual."
>
> p. 144

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Source: https://www.industrydocuments.ucsf.edu/docs/ppcl0000

Brown, S. M., et al., "Effect on Mortality of the 1974 Fuel Crisis," Nature 257: 306-307, September 25, 1975.

> "The 1974 fuel crisis was a natural experiment. It presented the opportunity to test the hypothesis that a decrease in vehicular exhaust fumes would have a beneficial effect on health. . . . Dramatic decreases were noted in death rates for several major categories of disease. . . .The disease showing the greatest relative change was chronic lung disease."
>
> p. 306

Sterling. T. D., "Additional Comments on the Critical Assessment of the Evidence Bearing on Smoking As the Cause of Lung Cancer: Communication from Sterling," American Journal of Public Health 66(2): 161-164, February, 1976.

> "[I]ncreases in lung cancer among women may well be associated with the increasing employment of women in occupations where they are exposed to physical and other stresses."
>
> p. 162

Smith, B., "Cancer Incidence," New Scientist, pp. 304-305, February 5, 1976.

> "The concentration on smoking as the sole cause of lung cancer simply discourages the search for other environmental factors. If asbestos did not cause a very unusual lung tumour, it might not have been picked up as the cause of the much commoner bronchial one."
>
> p. 305

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